NEUROPLASTICITY

The brain is constantly adapting throughout a lifetime, though sometimes over critical, genetically determined periods of time. Neuroplasticity is the brain's ability to create new neural pathways based on new experiences. It refers to changes in neural pathways and synapses that result from changes in behavior, environmental and neural processes, and changes resulting from bodily injury. Neuroplasticity has replaced the formerly held theory that the brain is a physiologically static organ, and explores how the brain changes throughout life.

Neuroplasticity occurs on a variety of levels, ranging from minute cellular changes resulting from learning to large-scale cortical remapping in response to injury. The role of neuroplasticity is widely recognized in healthy development, learning, memory, and recovery from brain damage. During most of the 20th century, the consensus among neuroscientists was that brain structure is relatively immutable after a critical period during early childhood. It is true that the brain is especially "plastic" during childhood's critical period, with new neural connections forming constantly. However, recent findings show that many aspects of the brain remain plastic even into adulthood.

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Plasticity can be demonstrated over the course of virtually any form of learning. For one to remember an experience, the circuitry of the brain must change. Learning takes place when there is either a change in the internal structure of neurons or a heightened number of synapses between neurons. Studies conducted using rats illustrate how the brain changes in response to experience: rats who lived in more enriched environments had larger neurons, more DNA and RNA, heavier cerebral cortices, and larger synapses compared to rats who lived in sparse environments.

A surprising consequence of neuroplasticity is that the brain activity associated with a given function can move to a different location; this can result from normal experience, and also occurs in the process of recovery from brain injury. In fact, neuroplasticity is the basis of goal-directed experiential therapeutic programs in rehabilitation after brain injury. For example, after a person is blinded in one eye, the part of the brain associated with processing input from that eye doesn't simply sit idle; it takes on new functions, perhaps processing visual input from the remaining eye or doing something else entirely. This is because while

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certain parts of the brain have a typical function, the brain can be “rewired”—all because of plasticity.

Neuroplastic Responses to Nervous System Damage ;

There are four major neuroplastic responses of the brain to damage:

1. Degeneration.
2. Regeneration.
3. Reorganization.
4. Recovery of function

1. Neural Degeneration

- Neural degeneration (neural deterioration) is a common component of both brain development and disease. A widely used method for the controlled study of neural degeneration is to cut the axons of neurons.
- Two kinds of neural degeneration ensue: anterograde degeneration and retrograde degeneration.

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1. Anterograde degeneration is the degeneration of the distal segment the segment of a cut axon between the cut and the synaptic terminals.
2. Retrograde degeneration is the degeneration of the proximal segment the segment of a cut axon between the cut and the cell body.

Anterograde degeneration occurs quickly following axotomy, because the cut separates the distal segment of the axon from the cell body, which is the metabolic center of the neuron. The entire distal segment becomes badly swollen within a few hours, and it breaks into fragments within a few days.

The course of retrograde degeneration is different; it progresses gradually back from the cut to the cell body. In about 2 or 3 days, major changes become apparent in the cell bodies of most axotomized neurons. These early cell body changes are either degenerative or regenerative in nature. Early degenerative changes
to the cell body (e.g., a decrease in size) suggest that the neuron will ultimately die usually by apoptosis but sometimes by necrosis or a combination of both.

Early regenerative changes (e.g., an increase in size) indicate that the cell body is involved in a massive synthesis of the proteins that will be used to replace the degenerated axon. But early regenerative changes in the cell body do not guarantee the long-term survival of the neuron; if the regenerating axon does not manage to make synaptic contact with an appropriate target,the neuron eventually dies. Sometimes, degeneration spreads from damaged neurons to neurons that are linked to them by synapses; this is called transneuronal degeneration. In some cases, transneuronal degeneration spreads from damaged neurons to the neurons on which they synapse; this is called anterograde transneuronal degeneration. And in some cases, it spreads from damaged neurons to the neurons that synapse on them; this is called retrograde transneuronal degeneration.

2. Neural Regeneration

Neural regeneration- the regrowth of damaged neurons does not proceed as successfully in mammals and other higher vertebrates as it does in most invertebrates and lower vertebrates.

The capacity for accurate axonal growth, which higher vertebrates possess during their early development, is lost once they reach maturity. Regeneration is virtually nonexistent in the CNS of adult mammals, and is at best a hit-or-miss affair in the PNS. In the mammalian PNS, regrowth from the proximal stump of a damaged nerve usually begins 2 or 3 days after axonal damage.

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There are three possibilities. First, if the original Schwann cell myelin sheaths remain intact, the regenerating peripheral axons grow through them to their original targets at a rate of a few millimeters per day. Second, if the peripheral nerve is severed and the cut ends become separated by a few millimeters, regenerating axon tips often grow into incorrect sheaths and are guided by them to incorrect destinations; that is why it is often difficult to regain the coordinated use of a limb affected by nerve damage even if there has been substantial regeneration. And third, if the cut ends of a severed mammalian peripheral nerve become widely separated or if a lengthy section of the nerve is damaged, there may be no meaningful regeneration at all; regenerating axon tips grow in a tangled mass around the proximal stump, and the neurons ultimately die.

Some CNS neurons are capable of regeneration if they are transplanted to the PNS, whereas some PNS neurons are not capable of regeneration if they are transplanted to the CNS. Clearly, there is something about the environment of the PNS that promotes regeneration and something about the environment of the CNS that does not. Schwann cells,

which myelinate PNS axons, clear the debris resulting from neural degeneration and promote regeneration in the mammalian PNS by producing both neurotrophic factors and cell-adhesion molecules (CAMs). The neurotrophic factors released by Schwann cells stimulate the growth of new axons, and the cell-adhesion molecules on the cell membranes of Schwann cells mark the paths along which regenerating PNS axons grow. In contrast, oligodendroglia, which myelinate CNS axons, do not clear debris or stimulate or guide regeneration; indeed, they release factors that actively block regeneration. Furthermore, oligodendroglia tends to survive for long periods of time (e.g., months) after nerve damage, thus chronically inhibiting regeneration of the axons.

If the factors that promote accurate regeneration in lower vertebrates can be identified and applied to the human brain, it might be possible to cure currently untreatable brain injuries. Remarkably, when invertebrates lose an entire limb, the regenerating axons release a factor that promotes regeneration of that limb.

3. Neural Reorganization:

Adult mammalian brains have the ability to reorganize themselves in response to experience.

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Most studies of neural reorganization following damage have focused on the sensory and motor cortex of laboratory animals. Sensory and motor cortex are ideally suited to the study of neural reorganization because of their topographic layout. The damage-induced reorganization of the primary sensory and motor cortex has been studied under two conditions: following damage to peripheral nerves and following damage to the cortical areas themselves.

The following studies have been particularly influential:

- Kaas and colleagues (1990) assessed the effect of making a small lesion in one retina and removing the other. Several months after the retinal lesion was made, primary visual cortex neurons that originally had receptive fields in the lesioned area of the retina were found to have receptive fields in the area of the retina next to the lesion; remarkably, this change began within minutes of the lesion.
- Sanes, Suner, and Donoghue (1990) transected the motor neurons that controlled the muscles of rat's vibrissae (whiskers). A few weeks later, stimulation of the area of motor cortex that had previously elicited vibrissae movement now activated other muscles of the face.

- Cortical Reorganization Following Damage in Humans:

Demonstrations of cortical reorganization in controlled experiments on nonhumans provided an incentive to search for similar effects in human clinical populations. One such line of research has used brain-imaging technology to study the cortices of blind individuals. The findings are consistent with the hypothesis that there is continuous competition for cortical space by functional circuits. Without visual input to the cortex, there is an expansion of auditory and somatosensory cortex, and auditory and somatosensory input is processed in formerly visual areas. There seems to be a functional consequence to this reorganization: Blind volunteers have demonstrated skills superior to those of sighted controls on a variety of auditory and somatosensory tasks.

Mechanisms of Neural Reorganization:

- Two kinds of mechanisms have been proposed to account for the reorganization of neural circuits: a strengthening of existing connections, possibly through release from inhibition, and the establishment of new connections by collateral sprouting.
- Indirect support for the first mechanism comes from two observations: Reorganization often occurs too quickly to be explained by neural growth, and rapid reorganization never involves changes of more than 2 millimeters of cortical surface.
- Indirect support for the second mechanism comes from the observation that the magnitude of long-term reorganization can be too great to be explained by changes in existing connections. Although sprouting and release from inhibition are considered to be the likely mechanisms of cortical reorganization following damage, these are not the only possibilities

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4. Recovery of Function after Brain Damage:

Recovery of function in humans after nervous system damage is a poorly understood phenomenon. Nevertheless, there is a general consensus that recovery of function is most likely when lesions are small and the patient is young.

Neuroplastic phenomena are presumed to underlie recovery of function. However, it has proven difficult to provide strong evidence for this assumption. Recovery of function after nervous system damage is difficult to study because there are other compensatory changes that can easily be confused with it.

Cognitive reserve (roughly equivalent to education and intelligence) is thought to play a role in the improvements observed after brain damage that do not result from true recovery of

brain function. Let me explain. Kapur (1997) conducted a biographical study of doctors and neuroscientists with brain damage, and he observed a surprising degree of what appeared to be cognitive recovery. His results suggested, however, that the observed improvement did not occur because these patients had actually recovered lost brain function but because their cognitive reserve allowed them to accomplish tasks in alternative ways.

Cognitive reserve has also been used to explain why educated people are less susceptible to the effects of aging-related brain deterioration. Several recent experiments in laboratory animals have shown that various treatments are maximally effective only if initiated within a few days of the brain damage. This finding has a potentially vital implication: It suggests that rehabilitation of human patients with brain damage should begin as soon as possible after the damage occurs.

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